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ART. I.—*On Functional Valvular Disorders of the Heart.* By J. M. DACOSTA, M. D., one of the Physicians to the Pennsylvania Hospital.

THE distinction between organic and functional cardiac affections is one practically of the highest importance, and, in regard to the differences established by their coarser features, is for the most part readily recognized. Yet every physician has constantly instances of doubt arising, and when he attempts to solve it in the light of generally existing knowledge will often be led into error. This is particularly prone to happen in the diagnosis of valvular disease, and depends chiefly on the significance attached to that landmark of affections of the valves—a murmur. In this paper I shall attempt to show what under certain circumstances its real value and meaning are, and shall bring together a series of observations commenced seven years ago, some of which will be reported in detail, while the bulk has been used to frame the general statements and deductions made.

It is scarcely necessary to premise that cardiac murmurs are usually of twofold kind: 1st. Dependent upon obvious and persistent organic disease; 2d. Owing to altered conditions of the blood. The former are well understood, and their character and significance are described at length in every treatise on practical medicine; the latter are chiefly those anæmic murmurs which have their seat ordinarily at the base of the heart, and occur with each contraction; and, although every now and then cases are met with in which they lead to the supposition of serious valvular trouble, yet their situation and systolic time, the coexistence with obvious signs of anæmia, and the normal size of the organ, form a combination of symptoms which, in the vast majority of instances, sufficiently distinguish them. But besides these two great groups, we find on the one hand cases of valvular disease without murmur, and on the other hand, cases in which a murmur in the heart exists without marked blood-change or constant lesion—from mere temporary disorder of the valvular apparatus. It is these that I intend more particularly to examine.

The question, then, that I propose to discuss is: Are there cardiac murmurs occurring in persons not anæmic, and, therefore, closely simulating organic valvular troubles; and again, what is the origin of such murmurs, and what means have we to prevent ourselves from being deceived?

Now let us take up the valves in order. Do such murmurs happen at the base? I have met with but few instances where an aortic murmur seemed to me to belong to the category alluded to, and there it was generated during active excitement of the heart, was soft, was not propagated into the arterial system, occurred with contraction of the ventricles, was followed by a distinct second sound, and did not last. At and near the orifice of the pulmonary artery, also, murmurs are found which are not associated with organic change of the valves or in the vessel, which may be due to constriction by surrounding altered pericardial or pulmonary structure, or again be inconstant and purely functional. In the former instance the more permanent sound coexists with the signs of either pericardial or pulmonary disease, and particularly of phthisis. In the latter there is no such combination and no such history. To cite a case in point:—

Case I.—William T., forty-one years of age, a patient in the Pennsylvania Hospital, who had had attacks of acute rheumatism. Subsequent to one of these, and while the action of the heart was very irregular, a blowing sound was perceived occurring with the systole. It was heard towards midsternum, but especially at the left base and at the second left cartilage, not on the right side at the aortic cartilage, and only very faintly at the apex. No murmur was present in the veins or arteries of the neck. The cardiac murmur gradually disappeared as the heart's action became more regular; for a time violent exercise still excited it, but finally the blowing sound ceased. The patient was about six weeks under observation. The murmur was clearly not anæmic. I admit that the fact that it happened in a rheumatic person suggests that it was the result of endocarditis; yet I do not adopt this view, because it was only marked with the irregular action of the heart, because its seat was not that of a murmur of acute endocarditis, because it occurred without any decided constitutional symptoms, and because as the heart became more regular and slower in its action it ceased.

Let us now turn to the auriculo-ventricular openings. As regards the tricuspid orifice, a murmur at or near the ensiform cartilage, therefore at the place where we clinically listen to the tricuspid valves, has never, in my experience, been of the origin under discussion, never at least in a healthy heart; though I know that it may happen in a dilated heart without valvular disease. But this is a part of the subject that we shall further on examine more in detail. With reference to a mitral murmur, namely, to a murmur at or near the apex beat, the phenomena in question are very usual;

indeed, it is there that they are pre-eminently met with. I will here bring forward some illustrative cases, selected almost at random from a considerable number.

Case II.—Charles S., an infantry soldier, at the U. S. Hospital, Turner's Lane, in May, 1864, for treatment for palpitation. This was one of those typical cases of irritable heart so common during the war. He never had had rheumatism, had indeed always enjoyed good health, until while in the army, he was attacked with fever, probably typhoid fever. On recovering from this and on returning to duty, he was much troubled with shortness of breath and beating at the heart, which symptoms were followed by sharp, though intermitting pain in the cardiac region. When first examined, the impulse was found to be somewhat forcible, and to occur one hundred and twelve times in the minute. The first sound was obscured above the apex by a soft murmur, which did not extend to the base, was indeed not heard over any other part of the chest. The man looked well, and there was not the slightest evidence of scurvy or anæmia.

In July, 1864, he was sent away with a squad of "emergency men," but did no duty, finding it impossible to walk any distance. I saw him again in August; he looked quite healthy, but was still troubled with palpitation; the impulse was about the same, and the cardiac murmur perceived, though not so distinctly. He remained under observation until Oct. 15th, the impulse retaining its force, and the pulse continuing above 100, but the murmur had disappeared; the most careful analysis with a double stethoscope failed to detect it; the first sound was noted to be heavier than normal.

Case III.—Isaac F., twenty-three years of age, also a soldier. He enlisted Jan. 19, 1864, for three years, and was well prior to enlistment. He was sent to his regiment on the 27th of April, having been detained at the rendezvous by an attack of typhoid fever. He was in two battles, and did duty with his regiment until May 12th, when difficulty in marching and excessive beating of the heart caused him to be sent to the hospital. While at the U. S. Army Hospital he had an attack of diarrhœa, accompanied by febrile symptoms; it was indeed his belief that it was another attack of fever. I saw him Aug. 18th; he did not look like a sick man, yet he evidently suffered a good deal. Pain in the back, dizziness and dimness of vision were complained of, and were found to be associated with a heart constantly beating 150 times in the minute. The impulse of the excited organ was extended, moderately forcible, the heart sounds were distinct, the first being obscured over the left ventricle, and above the apex, by a softish murmur occurring with the impulse; the second sound was very marked at the base both over the aortic and pulmonary artery cartilage. There was no hypertrophy—the transverse diameter of the percussion dulness was $3\frac{1}{2}$ inches, the perpendicular $3\frac{1}{8}$ inches. The man's height was 5 feet $10\frac{1}{2}$ inches, the girth of chest $33\frac{1}{4}$ inches. Corresponding closely to the outline of cardiac dulness, excepting just over the apex, was marked tenderness; pressure in fact made him feel faint. The pulse was feeble, but there were no murmurs or signs of disorder of the circulatory system besides those noted.

The murmur continued to be plainly noticed until September 22d, being always heard over the same part of the cardiac region, never at midsternum or at the second interspace on the right side. The man's condition improved very much, the dizziness and pain left, and he was able to sleep at night, no longer being disturbed, as at the onset of the case, by frightful dreams.

In October, the murmur disappeared entirely, was not audible even with a double stethoscope, though the impulse was still repeatedly observed to be 120, and the second sound remained very distinct, more distinct over the middle of sternum and to the right, than to the left, and over the pulmonary artery.

Case IV.—Ann D., forty years of age, a patient in the Pennsylvania Hospital, in 1866, admitted with bronchitis. While under treatment for this, it was noticed that the heart's action was excited and strong, and she stated that she was liable to prolonged attacks of palpitation; she was not anæmic. Several times during her stay in the Hospital, and while the heart was beating 118 times in the minute, a soft, systolic, blowing sound was noticed above the apex; the second sound of the heart was everywhere distinct. The murmur was only heard over the front of the chest, and became inaudible at the base of the heart. The pulse was at all times very compressible. The patient gradually recovered from her cough, and under cardiac sedatives the action of the heart became much slower. She was discharged April 10th, having been in the Hospital about two months; on several examinations prior to her discharge not a trace of the murmur could be detected.

Case V.—Mary K., seventeen years of age, was admitted into the Pennsylvania Hospital, in January, 1867. The May previous she had had tertian intermittent, and the chills, with occasional intermissions procured by the use of quinia, continued for more than two months. After they had ceased, she found herself weak and suffering with pains in her limbs, frequent headaches, and distressing palpitation of the heart upon the least exertion. Her appetite remained good; indeed she had on February the 8th so far regained her health, that she complained of nothing but the excessive cardiac action, and she had the appearance of a healthy girl, with cheeks, lips, and tongue of good colour. Her chest was well developed, and all her functions were regularly performed.

The impulse of the heart extended over two intercostal spaces, but was only of moderate strength; the transverse diameter of the cardiac dulness measured $3\frac{7}{8}$ inches; the vertical $3\frac{3}{4}$. To the left of the sternum, from the third to the second interspace, and with its point of greatest intensity at the third interspace, a soft, rather low-pitched murmur was found to accompany each beat of the heart. It was transmitted transversely to the left for about $1\frac{1}{2}$ inches, and more faintly upwards towards the cartilage of the pulmonary artery. It became intensified by active exercise, and it was then very evident that its point of greatest distinctness was in the third interspace.

At the apex, strictly speaking, no murmur was audible; the first sound there being dull, and heavier than usual, the second more distinct. At the ensiform cartilage the first sound was shorter and sharper than at the apex. The first sound over the aortic cartilage was short and sharp, but wholly free from murmur; the second aortic sound was very distinct, and more distinct than that of the pulmonary artery. No murmur was heard along the inner edge of the left scapula, and no abnormal sound in the vessels of the neck. The breathing was always rather rapid, 32 in the minute, and the pulse 117.

She took for a time quinia with strychnia and iron, but this was exchanged for digitalis—the effect of which was to quiet the rapid action of the heart and to reduce it to 97—and subsequently for aconite. But her treatment was much broken in upon by the necessity of constantly prescribing for violent headaches, to which she was very liable. The aconite, however, seemed to have some influence on these, and it diminished the force as well as the frequency of the heart's action; the impulse being repeatedly noted to be 86. The blowing sound became very indistinct, though it still could be perceived when the circulation was quickened by excitement or exercise, up to the period she left the Hospital, the last week in April. Her appearance at the time was that of perfect health.

The general character of the murmur, and the attending phenomena may be learned from these cases. But let us analyze its traits.

In calling attention to it in my work on *Medical Diagnosis*, I have described it "as much more likely to be heard at the apex, or rather, according to my own observations, somewhat above the apex, than is a murmur owing to changes in the blood; and it differs from the systolic blowing sound of mitral disease partly by the peculiarity of seat just mentioned, partly by its non-diffusion, its absence at the back of the chest, and the very much more general want of harshness in the inconstant murmur." Dr. James Andrew has published in *St. Bartholomew's Hospital Reports*, an able paper on endocardial murmurs, and while discussing these systolic blowing sounds reaches conclusions which are substantially the same, and lays, moreover, stress on the want of intensification of the normal second sound of the pulmonary artery. I have now before me the notes of about fifty cases I have collected, and am able in a general way to subscribe to the correctness of this opinion. I find, indeed, in a number of my observations, the statement that the second aortic sound, listened to at the second cartilage on the right side at its junction with the sternum, was clearer and better marked than the second sound of the pulmonary artery, or that heard at a corresponding situation on the left side or over the second interspace. But I also find in a few the reverse; and, moreover, when we take into account that during excited action of the heart—the state that usually accompanies these murmurs—the sounds at the base are very apt to become sharper and more distinct, we can understand how difficult comparative observations are. Setting aside the question whether cases of organic mitral disease are always accompanied by an intensification of the second sound of the pulmonary artery, we perceive, then, these inorganic cardiac murmurs occurring under circumstances which often render the recognition of a comparatively accentuated second sound an uncertain matter.

The absence of the murmur at the back, not finding it, for instance, at or near the lower angle of the left scapula, where a murmur from organic, mitral regurgitation is so common, is a more valuable sign. Yet here, too, we encounter exceptions. On the one hand, a murmur not dependent on organic changes may be there perceptible. On the other, we know that all mitral organic murmurs are not propagated to the left axilla, and to the back; hence not finding a murmur there, is, though strong evidence, not positive evidence of the murmur heard over the cardiac region not being due to structural disease of the mitral valve. Intensity of sound is, I believe, necessary to the murmur at the back being audible; and this intensity is in rare cases of mitral disease wanting. But the want of intensity which inorganic murmurs usually possess explains why they are, as a rule, not propagated beyond the cardiac limits.

A more certain sign, I think, than either of the two mentioned, is the localization of the murmur. The more carefully I have kept my notes the more distinctly I find it stated, that it is not an apex murmur, strictly speaking, but rather occurs above the apex, more over the body of the left ventricle. The third interspace is the point at which it is very apt to be most marked; from there it extends faintly to the second interspace on the left side, in some cases distinctly enough to make one believe in its seat being the pulmonary artery. It may occupy the whole of the first sound, but it more commonly does not, and at the apex this, or a portion at least of this, is almost always to be detected. In only about the proportion of three instances out of fifty did I find that it was audible at the right side of the sternum towards the aortic cartilage, and in one of these it was only audible with a double stethoscope. But even when it can be perceived at the right base, it is always there very faint, and we are never in doubt that this is not the place of its intensity.

It is always synchronous with the first sound or impulse, and never passes into the second sound. In character it is generally soft, certainly devoid of harshness, and not very loud. It has in the vast majority of instances a low pitch.

The above are its prominent traits, but there are a few more points worthy of notice which it presents, and which I have studied in connection with it; and one of these is that in some cases it is very much influenced by pressure. I attend, for instance, a lady, about thirty years of age, suffering from chronic intestinal disease. There is no organic cardiac affection, for there are neither symptoms nor physical signs indicating it, but the heart's action is generally rapid, and sometimes she has decided attacks of palpitation. During these, or indeed whenever the action of the heart is more than usually quick, a softish systolic murmur above the apex is discernible, and on firm pressure with the stethoscope, or even with the ear, it changes at once into a much louder, harsher sound, taking up then the whole of the first sound, both near and at the apex. During the ordinary state of her circulation not the slightest murmur is perceptible.

Another point which I have studied in connection with these endocardial blowing sounds, is the manner in which the respiration affects them. The record of a few cases may be adduced in proof.

Case VI.—Margaret S., a patient in the Pennsylvania Hospital in April, 1861, admitted for pain in the back and dysmenorrhœa. Her general health was fair; she was not anæmic, and did not suffer from any pulmonary or cardiac disease; yet the respirations were 28, the pulse beat 96 times in the minute; and accompanying the constantly excited action of the heart there was, above the apex and also at the left base, a soft systolic murmur, which became inaudible at the right edge of the sternum. It was not found in the left axilla, though part of the first sound of the heart was there heard; nor was it to be perceived in the inter-scapular space on the left side even with a double stethoscope. The second sound of the heart was met with at the back, as a very distinct sound; and with reference to the sound in front, it was observed to be very clearly marked, the second aortic being more sharply defined than the second sound of the pulmonary artery. The cardiac percussion dulness was normal; the impulse of average strength, and slightly extended. There was no murmur in the carotids; at times a very slight venous hum was noticed.

Exercise made the murmur in the heart more distinct; it also became so during full-held expiration; it disappeared, a peculiar clacking sound taking its place, during full-held inspiration.

Case VII.—John S., forty-three years of age, a tuberculous patient at the Pennsylvania Hospital. The tubercle affected a considerable portion of the left lung, and both apices. The heart's action was rather forcible; the percussion dulness, though large, did not extend beyond midsternum or past the nipple. The first sound at the apex was dull and murmurish, but a distinct murmur was only excited when the action of the heart was much quickened by exercise. The murmur had its seat above the apex, was inaudible at the base, and limited to the cardiac region, not a trace of it being found in the left axilla, or posteriorly. Forced breathing, just as exercise, developed a murmur; this was particularly the case during full-held inspiration, which at the same time increased the second sound of the pulmonary artery; held expiration gave uncertain results. It did not seem to produce the murmur, but it caused so much irregularity of action as to make the observation doubtful. Only a very slight irregularity happened during held inspiration; and as the heart's action quieted down the murmur disappeared.

From these observations it is evident that the murmur is much modified by the respiratory acts. Whether inspiration or expiration modifies it most, and how the results obtained compare with trials on organic murmurs, is a matter that I shall not here further inquire into. I will merely say that these functional murmurs have seemed to me to be far more influenced.

When now we turn to a more general survey of the phenomena with which these physical signs are associated, we find that they occur in persons who present palpitation and more or less shortness of breath as symptoms, but not that violent dyspnœa which we encounter in some organic valvular affections; nor is dropsy a concomitant. Anæmia may or may not be witnessed, but is generally not; it is, in fact, rather to be viewed as a complication than as a causing element. Thus, almost all the cases from which I have made these deductions were healthy-looking persons, and showed neither in their colour nor on examining the vessels of the neck any evidence of deteriorated blood. But very nearly all the cases that I have seen have been those in which the heart's action was frequent, and in many it was also irregular. In a few it was irregular without being frequent.

The greater the rapidity with which the heart beats, the more apt are we, I think, to have the murmur engendered. But we cannot fix upon any limit which is sure to produce it. Thus, it became audible in one patient when his heart was beating 140, and disappeared when the action was reduced to 120. In another patient the murmur was heard with the cardiac impulse at 100, was not audible at 84, and the man, who was under observation for three months, left the hospital with a pulse of 70, and without any abnormal sound being recognizable.

Besides frequency, irregularity is stated to be a causing element. The murmur may occur while the heart is acting irregularly, or it may happen in contractions subsequent to the irregularity.

The pulse in cases in which the murmur is encountered, is, as a rule, quick but not full. I have taken several sphygmographic records in those presenting these murmurs, and I find the traces not very ample, the line of ascent nearly vertical, the summit broad, and the line of descent very undulatory. The traces differ very decidedly from the great, almost characteristic, irregularity of mitral lesions. Were it not that in some of the cases of functional murmur, we have, as already mentioned, irregular cardiac movement, the tracing might become a valuable diagnostic element, and even as it is, such marked irregularity does not occur as in organic disease, at least, I have not as yet met with it in any case examined.

The maladies with which we find associated the state of the heart giving rise to a functional murmur, are all those in which deranged action of the organ is prone to occur sympathetically, as disorders of digestion, or of the uterus. Again, we perceive it where the signs of disturbed function seem to constitute the complaint, and in which no other than cardiac symptoms are present. Or, again, I have met with this murmur in perverted innervation of the heart in diseases of the brain; and it has been for some time a question in my mind whether the cardiac murmurs in fevers—I do not mean those that occur at the base—are not really of the kind under consideration, and not blood murmurs, as generally supposed. Whether the murmurs of chorea belong to the class we are investigating, I do not care here to discuss, since this would involve the mooting of the whole question of the relation of this strange nervous affection to rheumatism and rheumatic endocarditis. Some of the cases certainly, those in which the murmur disappears, are similar. In diphtheritic paralysis I have encountered these inorganic murmurs, and that in instances in which no signs of anæmia were any longer present.

Studying the influence of the respiration on these murmurs unassociated with valvular lesions, caused me to inquire into their prevalence in diseases of the lungs. In consequence I have been led to observe their frequent combination with those respiratory affections in which decided obstruction in the pulmonary circulation occurs. Thus, in certain instances of extensive tubercular infiltration, in many cases of asthma, and in a fair proportion of cases of pneumonia, a mitral murmur, possessing all the characters mentioned in this paper, is met with. And I have encountered it in pleuritic effusion, where the patient dying from an abdominal disease, an opportunity for inspecting the valves of the heart showed them to be sound, and the organ itself neither hypertrophied nor dilated.

Excepting in these pulmonary cases, the murmur has been described in the foregoing pages as linked to more or less persistent symptoms of cardiac disturbance and as tolerably constant. But I may here state, that it also happens in perfectly healthy persons during temporary excitement of the heart, and that then it does not recur excepting with excitement. For instance:—

Case VIII.—Mr. B. consulted me in February, 1868, on account of a bronchitis which he thought was kept up by living on the Alleghany Mountains. The affection was trifling, consisted, indeed, of but little more than a slight expectoration in the morning, which was apt to cease in the summer and reappear in the winter, and was not associated with the slightest difficulty in respiration. Mr. B. was a vigorous man in the prime of life, of ruddy countenance, and with a splendidly developed chest. While examining him, I noticed the rather rapid beating of the heart, and on causing this to be increased by letting him walk very quickly up and down the room, heard towards the apex a soft systolic murmur which was nowhere else distinct over the cardiac region, was inaudible at the base, and did not completely take the place of the first sound. The murmur was also heard, though very faintly, near the inferior angle of the scapula. Never, in the history of the case, had there been rheumatism, or attacks of palpitation, or any signs of cardiac difficulty.

It may be objected to this case, that being seen but once, no proof was afforded that the murmur really ceased, and only occurred during increased cardiac action. But I have examined a number of similar cases against which this objection cannot be urged, and which, when the heart was not excited, did not present a trace of murmur or any signs of an abnormal cardiac state. Thus, in one person I heard a distinct blowing sound when the heart was beating quickly on December 13th, and January 13th, but none during quieter action of the organ on February 1st, March 2d, or April 3d. These temporary murmurs have all the characteristics of those discussed. And I would particularly call the attention of examiners for life insurances and of surgeons inspecting recruits or discharging soldiers to them. I know that they are constantly mistaken for signs of valvular disease, and during the war a number of soldiers got their discharge who turned out to have merely murmurs of this kind. Nor are murmurs from mere temporary functional excitement of the heart uncommon; indeed, estimating roughly, I should say, that such a murmur can be produced in at least one out of twenty healthy persons, and I am inclined to put down the proportion as much larger.

These remarks tend to shake somewhat rudely our faith in the value of murmurs as signs of chronic valvular disease. And so they do, if we merely look to the fact of the occurrence of the murmur without taking its characters into account. In truth, the whole inquiry shows that we cannot disregard so entirely the qualities of a murmur as has been done of late years, and that irrespective of its time of occurrence and position, its harshness or softness, its duration and pitch, should be studied. This inquiry further shows how utterly fallacious it is to be satisfied from one or two examinations that a man has a valvular disease of the heart, simply because with signs of functional disturbance of the organ he presents a cardiac murmur which we find not to be anæmic. And lastly, it shows what estimate we are to place on the cases of valvular disease that have said to have been cured, on the evidence that the blowing sound heard over the heart had disappeared.

While dwelling on the characters of the sound as significant, and calling attention to the mistakes that may happen from not considering these, I shall, even at the risk of some repetition, point out the distinguishing features of the functional disorder from those which it most resembles.

And first, from mitral regurgitation. In this the persistency of the murmur, its occupying the whole of the first sound, its diffusion over the cardiac region, its point of intensity more strictly at the apex, its usually harsher character and higher pitch, are of chief importance. To these we may add as valuable, though not as positively distinctive elements, the fact of the murmur being heard posteriorly and the accentuation of the second sound of the pulmonary artery. Often too we have the greater dyspnœa and the tendency to dropsy. I have purposely not as yet stated that we can lay stress on the physical signs of hypertrophy of the heart, for we shall presently see that with these murmurs not significant of organic valvular disease, enlargement of the heart may be present. Still, in the great majority of cases, they are, undoubtedly, not so combined; and if, with the extended impulse, we find decided increase in the cardiac percussion dulness, we can usually attach importance to it. We must, however, be careful to ascertain the signs accurately, and not to confound the augmented impulse with, perhaps, the rather more evident dulness on percussion of persistent palpitation with the manifestations of true enlargement.

How necessary it is to take all these elements into account, and how apt we are to fall into error, may be learned from two cases which came under my observation during the past year, and in both of which the disappearance of the murmur placed the diagnosis beyond doubt. One happened in a clergyman, who, examined by three physicians—all of them men of great distinction—at different times, was stated by one to have a mitral lesion, by the second, uncomplicated hypertrophy of the heart, and by the third, no cardiac affection of any kind. The murmur was soft, systolic, and coexisted with throbbing action of the heart and slightly increased dulness on percussion. Similar phenomena, only that any approach to hypertrophy was absent, were present in the second case, which had been pronounced by an expert in diagnosis to be one of very serious mitral disease.

Murmurs within the heart are, it is well known, not uncommonly due to altered conditions of the blood, particularly to anæmia. But these murmurs are found over the aorta or over the pulmonary artery, and not at or near the apex. Moreover, my experience regarding them is the same as that of Dr. Walshe. I have never met with them where there was not venous hum or a blowing sound in the arteries of the neck. Thus, their seat, and especially their diffusion, present differences. Besides, we have the looks of the patient and the history of the case. Yet here we find that sometimes the same circumstances which may occasion the one kind of murmur have occasioned the other. In truth, I have in a few instances found them combined, though I have omitted these instances in arriving at the deductions made in this essay.

The closest similarity to the inorganic murmurs near the apex is presented by certain rare cases of pericarditis. The exocardial sound may simulate most closely this, as it does every other variety of endocardial sound. I had a case under my charge at the Hospital in which every trait of the murmur we have here studied was present, excepting that it was more readily influenced by pressure than this commonly is. Nothing showed its true nature but an attack of pericardial inflammation with friction at the base, subsequent to which the seeming blowing sound in the neighbourhood of the apex was altered.

Before continuing now in the line of clinical investigation we have started on, it will serve our purposes best to inquire into the mode of production of the inorganic murmur, the attendant features of which we have been trying to fix. Leaving out for the present those infrequent cases in which a murmur of the kind, and not anæmic, is met with at the base of the heart, we find that it is most likely to be generated at the auriculo-ventricular orifice on the left side; we find, moreover, that it is unconnected with roughening of the ventricle or of the valves, in fact with structural change in either. The proof of the former is afforded by its site, which clinically corresponds more closely to the mitral orifice than to any other part; and this seems to me, too, to be a strong point against supposing the blowing sound to be due to tricuspid regurgitation; a view of the case which the known "safety valve function" of the tricuspid valve would at once suggest. The absence, further, of anything like fulness of the veins of the neck may be here adduced. That the murmur is inorganic, and not connected with persistent textural alteration, is proved by the readiness with which it appears and disappears, and by the results of post-mortem examinations. The valves were healthy in the autopsy briefly referred to in this paper; they were found to be healthy in other instances; and in analyzing the cases that I have seen here and there reported, with more or less amazement, as supposed disease of the mitral valve in which the inspection after death showed no disease, I identify most as belonging to the group I am endeavouring to elucidate, and am, I think, justified in accepting the facts to support my argument. Moreover, as regards a morbid state of the endocardium lining the muscular structure of the ventricle, we know that when this exists, no murmur, save under the rarest and most exceptionable circumstances, is generated.

If thus the murmur occurs at the mitral orifice, if it is unconnected with vegetations or other persistent lesion, how is it engendered? Here several solutions offer themselves; not one of them claims such superiority over the other as to admit of instant adoption. In truth, we are at once forcibly reminded of the opinion of one of the most ingenious experimentalists who has ever occupied himself with the subject. "No one," says Marey, "has up to the present time given a really scientific demonstration of the causes of blowing sounds."

The first solution which offers itself is, that there is an interference with the closure of the valves, permitting thus of temporary regurgitation. And the incompetency may be either owing to irregular contraction of the papillary muscles—a cause that corresponds with the spasmodic contraction of the cardiac muscles, which, according to Laennec, is the source of all cardiac murmur; or it may be due, as the experiments of Andrew indicate, to changes which happen at the mitral orifice. This, namely, is rendered round instead of remaining oval during the systole, and is, therefore, not closed by the cusps of the valves, which are adapted to the oval form. Or, again, the muscular fibres which the valves have been demonstrated to contain, may share in the palpitation, and thus, by their abnormal action, prevent perfect closure and allow regurgitation. If we are to adopt the view of regurgitation at all, this explanation seems to me the most likely; for the second is not yet sufficiently demonstrated, and the first is unsatisfactory on account of the difficulty of understanding how and why, excepting, perhaps, in cases of chorea, the papillary muscles should act spasmodically when the rest of the cardiac muscles do not.

In another solution of the question we need not assume regurgitation at all. We know that any flexible solid brought from a state of relaxation suddenly to a state of tension, vibrates, and this fact Rouanet and Bryan have made use of to defend the valvular theory of the first sound. We also know that changing the tension has an influence in changing the vibrations. If, further, we adopt the able observations of Chauveau, which prove that a vibrating current of fluid is the source of murmur; and supplement these observations by the ingenious experiments of Bergeon (*Des Causes du Bruit de Souffle*, Paris, 1868), which show that a sudden stoppage or shock produces vibrations in the surrounding fluid in consequence of the compression and reaction of the molecules;—we have all the elements which would explain how the murmur arises. I advance this theory as one to be considered. But the difficulties of the subject are so great that I have not myself as yet definitely adopted it. I will merely add that it is quite as consistent with facts as the theory of temporary regurgitation with misdirection of the current; and that it receives support from an experiment of Potain (*Gazette des Hôpitaux*, Mai, 1867), which proved that increasing the pressure and the rapidity of circulation of the same liquids, produced murmurs when these were otherwise not caused, and that they were the more readily generated the thinner the fluid. In this we would find the reason why the murmurs under consideration are audible for the most part only in an excited state of the circulation, and how palpitation is the more apt to occasion them when the composition of the blood is at the same time altered, which—from the character of the disorders with which we have seen the inorganic murmur to be associated—it frequently is, if even but slightly.

Perhaps the explanation here given applies to the murmur with rapidly acting, yet regular heart; the first, or that of temporary regurgitation, to the cases in which irregularity of the heart exists. I see, indeed, nothing far-fetched in adopting these different views for different cases. I contend that we are not bound to admit the idea of mitral insufficiency as the only one to account for phenomena which are otherwise explicable. Nay, when we consider how quickly some of these murmurs can be excited, how transitory they are, how they may be developed in healthy persons, and how close the coaptation of the mitral valves, we may well refuse to adopt the idea of mitral regurgitation for this whole class of inorganic murmurs. But why should not the altered tension of the valve which we have named as among the causes, be also produced by irregular muscular contraction, and then there be sufficient derangement to occasion regurgitation. Might not, in truth, even in cases in which irregularity does not exist, a constantly altered tension become associated with changed condition of the muscular fibres of the valves, either as the result of persistently deranged function, or from the same causes acting on their nutrition which are modifying the nutrition of the entire organ, indeed of the whole system? Thus, then, altered tension may be the beginning, and pass on into a state of things favouring valvular incompetency.

The inorganic cardiac murmur, near the apex, met with in obstructive diseases of the lung, is probably due to the same elements we have been considering, of increased action, altered valvular tension, and some change in the composition of the blood. To suppose the blowing sounds—certainly those in acute cases—to be due to mitral regurgitation, makes them very difficult of explanation. We might, indeed, assume that diminished supply of blood flowing into the left auricle with altered contraction of this part of the heart was the cause, but this supposition does not help us much, since the murmur is systolic and occurs when the valves are floated out to close the aperture; unless, indeed, we are prepared to adopt the opinion of Baumgarten, that the valves are closed by the auricular systole just prior to the contraction of the ventricles.

The occasional inorganic murmurs at the base, alluded to in the first pages of this paper, are explicable by remembering the altered rapidity with which the blood flows through the arterial openings into the great vessels, and the changed tension of their coats associated with the abnormal action of the heart.

But to return to the clinical bearings of the question, and particularly with reference to the murmur near the apex. This much is certain, that whether with or without regurgitation as the causing element, we must recognize murmurs significant of functional valvular disorder. In the cases which we have been thus far considering, the valvular derangement was nearly exclusively combined with mere functional affection of the heart. But may we have functional valvular trouble associated with organic disease of the walls and cavities? We certainly may, as these cases show:—

Case IX.—William H. S., twenty-one years of age, a soldier, who had enlisted in 1862 while in perfect health. He was attacked when in the field with diarrhœa, and on returning to duty, began to suffer from palpitation and difficulty of breathing; a cough, too, annoyed him; and partly on account of these symptoms, and partly in consequence of persistent irritability of the intestinal canal, he was sent away from the regiment and transferred to a military hospital in Philadelphia.

The impulse of the heart was extended and forcible, the transverse diameter four inches and a half; the pulse was compressible, and about 100. There was no swelling of the feet, and the man did not appear at all anæmic.

After he had been a short time under observation a systolic murmur above the apex was noticed. It was not diffused beyond the cardiac region, and, indeed, almost limited to the point mentioned. The murmur became most evident when the action of the heart was excited, and ultimately it was only evident under these circumstances. The man was for five months under observation, and no change in the character of the inconstant sound took place. Just prior to his discharge it was not heard.

Case X.—George S., a soldier, with hypertrophy of the heart, which was evidently developed while in the service. He never had acute rheumatism, and there were no signs of scurvy. Examined in March, 1864, the impulse was found to be extended and forcible, the cardiac percussion dulness increased, and a systolic murmur above the apex, and feebly transmitted to the left base was heard. The murmur was soft, and limited to the cardiac region.

Examined in October, the murmur was still audible.

A note taken in June, 1865, speaks of it as being yet perceptible. The impulse at that time was 88; the pulse regular, of good volume, the dilated hypertrophy undoubted; there was no dropsy. The murmur was not harsh, was most distinct near the left edge of the sternum at the fourth rib and in the third interspace, was faintly heard in the second interspace on the left side, and barely discernible at the aortic cartilage. The second aortic sound was far more distinct than the second sound of the pulmonary artery. At the apex proper the first sound of the heart was dull and heavy.

Case XI.—Jefferson H., twenty years of age, also a soldier. His history showed that he had had two attacks of fever; the last one, which he had shortly after the middle of February, 1864, was followed, on his return to duty, by constant palpitation, and by pain in the cardiac region, which he described as jerking. The palpitations were very severe at night, and he was obliged to sleep sitting in a chair, being much troubled with shortness of breath. He had a short cough, and once expectorated blood.

The impulse of the heart was forcible, extended, rapid; the cardiac percussion dulness large. A systolic murmur was noticed at several examinations, though it is not mentioned as constant, and when the action of the heart was very frequent—it often reached 140 in the minute—the blowing sound is described as heard only to the left of the sternum, and as having its seat of maximum intensity near the edge of the sternum in the third interspace, as faint in the second, and as scarcely perceptible at the apex. The examination was made with a double stethoscope.

In one of these patients, the murmur was very constant; in the others, it was very commonly found; in all, the heart's action was frequent. But I have met with cases of hypertrophy, or dilatation, in which the murmur only occurred during excitement of the circulation, or in which, as the frequency of the impulse became more normal, though the signs of enlargement remained, the murmur permanently disappeared. I have also known a murmur in an hypertrophied heart to cease when the organ had been quieted by aconite. With reference to the character of the murmur, I find on analyzing my cases, that it is precisely similar to the one I have already fully described in connection with mere functional disorder—similar in seat, in character, in not taking the place of the first sound completely, in want of diffusion; I am only inclined to lay more stress on the absence of accentuation of the second sound of the pulmonary artery. As regards the connection with irregularity of action of the heart, the murmur in enlarged hearts may occur during or subsequent to such irregular action, without, as I have had opportunities of satisfying myself, betokening organic valvular disease. I mention this particularly, because it has a distinct diagnostic bearing on cases in which a presystolic mitral murmur occurs, which we know is often not very marked, exhibits great variability, and frequently coexists with a heart very much disturbed in its rhythm.

Now it is true that this kind of murmur, which in the vast majority of instances is associated with a contracted mitral orifice, takes place just before the systole, and that the inorganic murmur happens with it; but every auscultator knows that where irregularity is present, this difference is very difficult, nay, may be impossible to appreciate. Hence it might be anything but an easy task to distinguish a case of most serious valvular lesion from one in which the valves are healthy. Nay, it may not be possible to make the diagnosis at first sight, and only by careful weighing of all the symptoms, particularly of the amount of dyspnœa, of the extent of irregularity, and of the history of the case, can we, in an irregularly acting heart, make out the true meaning of a murmur at or near the seat of mitral valve.

While discussing the murmur of functional kind without accompanying enlargement, I spoke of its connection with obstructive disease of the lung. The same connection may happen in instances of altered size of the heart, and make the similarity to persistent mitral regurgitation a very close one. Thus I met, at the Pennsylvania Hospital, with this case:—

Case XII.—P. S., fifty years of age, was admitted into the Hospital on the 15th of December, with difficult breathing and intense congestion of the lungs. The symptoms had existed only for four weeks, had come on in the midst of health, and had been followed by swelling of the feet, legs, and abdomen. The urine was scanty, contained albumen, about one-third of the test tube full, and transparent and granular casts and renal cells. The temperature was $101\frac{2}{5}^\circ$; the pulse ranged from 96 to 112, was compressible, and some beats were fuller than others. The lips were livid; the respiration was very much embarrassed, the man unable to breathe, except when propped up; loud, dry, and moist râles were heard over the whole chest, but there was no dulness on percussion over the lungs.

The cardiac impulse was extended, rather feeble compared with its extent, or with the increased percussion dulness. The first sound was indistinct and short, and near the apex a softish, systolic murmur was heard, not wholly replacing it; the second sound was everywhere long and distinct, seemed particularly so at the apex, but was not more so at the cartilage of the pulmonary artery than elsewhere. There was neither pulsation nor particular fulness of the veins of the neck; and the murmur was limited to the cardiac region, its point of intensity always being just near the apex.

Under dry cupping, both of the chest and over the kidneys, solution of acetate of ammonia, sweet spirits of nitre and other diuretics, quinia, a moderate amount of stimulus, and an occasional expectorant mixture of muriate of ammonia, the dropsical symptoms gradually disappeared, and the congestion of the lungs steadily decreased.

On the 30th of December the pulse was 90; the respiration 30; the temperature $98\frac{2}{5}^\circ$.

On the 4th of January the dropsy had nearly gone; but very few epithelial and granular casts were found in the urine, and only a trace of albumen. There were still some subcrepitant râles in the chest, but the breathing was not oppressed. The heart's action was regular, pulse fuller, so was the first sound, and the murmur was so indistinct that its existence was doubtful.

A note taken on the 28th, describes the man as convalescent and walking about the ward, without any dropsical symptoms. The cardiac signs were the same as regards the percussion dulness, the impulse seemed stronger. The first sound had still somewhat of the character of the second; but not a trace of murmur was discernible. Exertion, such as walking as fast as he could, did not produce it, though it caused slight dyspnœa. There was only a trifling cough remaining.

Here, then, was a case evidently of renal dropsy, either wholly acute, or engrafted on a previous renal trouble, and coexisting with cardiac enlargement and with embarrassment of breathing, during which a cardiac murmur was audible, which as the respiration became freer passed away. The fact that the murmur was associated with a dilated heart rather than with a hypertrophied one, is not of any consequence as regards the argument; for these functional murmurs occur alike in hypertrophy and dilatation, and are the same in both states, perhaps a little more evident in the former than in the latter, but otherwise presenting the same characteristics.

Cases of the kind are of importance to understand, and when I look in their light on instances that have been published of mitral regurgitation without mitral disease being found after death in which marked lung symptoms were present—and I may in passing, refer to an admirably reported case by Dr. Cuming in the number of the *Dublin Quarterly* for May, 1868—I derive from them a different impression. It is not the murmur in the heart, I think, that is the sign of the disease of the heart which has occasioned the pulmonary disorder; but the pulmonary disorder has given rise to the cardiac murmur which is looked upon as its determining cause.

The inorganic murmur, indeed, in cases attending cardiac enlargement admits usually of the same explanation as when it is associated with mere functional cardiac derangement. It is true that there may be in organic changes in the walls or cavities other causes at work which at times occasion a murmur that is not associated with lesion of the valve texture itself. There may be dilatation of the cavity gradually producing incompetency of the seemingly healthy valve; or, again, as suggested by Dr. Bristowe in a paper on mitral regurgitation which has become classical, the musculi papillares and chordæ tendineæ may not keep pace with the growth of the ventricular cavities. Yet this disproportion was only marked in three out of the six cases he describes; and these we might well admit as cases of organic valvular disease. The other three presented no such conditions; but unfortunately in all, the details as to the murmur and the other auscultatory phenomena are so meagre as to make it impossible to use them for purposes of differential diagnosis.

I believe, then, that the large number of cases of inorganic murmur, with hypertrophy or dilatation, are not associated with any kind of persistent alteration. The abnormal sounds are, for the most part, rapidly produced and disappear as rapidly. They have the same characters, and own, in by far the majority of instances, the same cause as the murmurs associated with mere functional disorder; in other words, they belong to and tend to prove the same law which I have been endeavouring in these pages to explain. Most probably, too, the apex murmur that is heard in some cases of soft, flabby heart, without valvular affection, is of kindred kind; but my observations on this point are as yet not enough to speak positively.

The murmurs, of the origin under discussion, when joined to hypertrophy or dilatation, are as rarely basic as they are when occurring with simple functional derangement. When they thus happen, their association with very frequent or irregular action of the heart, the absence of anæmic sounds in the neck, their temporary nature and want of harshness; and, on the other hand, a history of the case which would suggest a cause for a valvular trouble, and show the more special symptoms of this, or the exclusion of disease of the lung which by pressure would cause a narrowing of calibre—must, as in the purely functional cases, serve as diagnostic marks.

I shall, in conclusion, briefly moot a point of the utmost practical importance. Do cases of functional valvular disorder ever pass into organic valvular disease? This is a very difficult matter to settle, one, indeed, in which demonstration ceases, and belief begins; and I believe that it is so. I have seen cases, few it is true, that had the inconstant murmurs, the examination of which has occupied so large a share of this inquiry, and in which when I encountered them after the lapse of time, obvious valvular disease existed. But in every one the enlargement of the heart, which at first was slight, had become a marked feature. Hence, a case might begin with functional valvular disorder attending functional affection of the organ; hypertrophy may, as I know to have happened in many instances which I have had an opportunity of examining, follow the functional affection, and if this become marked, valvular disease may ensue. Such I believe to be a possible result, and while it is not susceptible of proof—for it may be suggested that the signs of valvular affection were from the outset due to a slight but increasing organic lesion—repeated examination of the question, and a careful weighing of the facts, in some of the cases I have seen, particularly as to their history, have caused my belief to become a firm conviction. If this sequence be true, how important a lesson it inculcates as to the importance of not neglecting functional affections of the heart; how it teaches us to treat that which if we make light of may pass from a curable into an incurable malady; how markedly it points to the means which will give the agitated organ rest as alike of value in the present and as preventive of mischief in the future.